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## Low Heart Rate Variability in Healthy Young Adult Males

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### 1. Introduction

The autonomic nervous system coordinates a person's responsiveness to physiological and environmental stressors. Attenuated respiratory sinus arrhythmia (RSA) with small standard deviation of the normal-normal electrocardiogram RR intervals (SDNN) is found in subjects with low heart rate variability (HRV) by time domain analysis (Montano et al., 2009). Low HRV is also characterized by sympathovagal balance shifted toward sympathetic predominance observed as an increase in the low frequency/high frequency ratio by frequency domain (fast Fourier transform) spectral analysis (Montano et al., 2009). However, inter- and intra-individual spectral changes are highly variable (Taverner et al., 1996) and the physiological significance is not always clear. Paced breathing at 0.2 Hz normally shifts sympathovagal balance toward greater vagal and less sympathetic activity (Driscoll and Diccio, 2000) because of increased tidal volume and/or minute ventilation (Pinna et al., 2006). Sympathovagal imbalance related to low HRV is a risk factor for hypertension and various other cardiovascular and non-cardiovascular diseases (Kuch et al., 2001; Montano et al., 2009). Epidemiological studies estimate that the prevalence of hypertension ranges from 8% in an urban adolescent population (Rabinowitz et al., 1993) to 43% in black physicians twenty-two years after medical school (Gillum, 1999). However, no studies have determined the incidence and behavioral significance of sympathovagal imbalance in healthy young adult African-Americans. Abnormal autonomic responsiveness to environmental stressors is thought to be an important factor in the evolution of essential hypertension (Fauvel et al., 1996; Mezzacappa et al., 2001; Lucini et al., 2002a; 2002b) and African-American males are a subpopulation at high risk for such hypertension (Gillum, 1979; Kaplan, 1994; Gillum, 1999). The present study was, therefore, designed to determine whether sympathovagal imbalance related to low HRV occurs in a population of healthy young adult African-American males and whether it is an indicator of abnormal responsiveness to environmental stressors.

### 2. Materials and methods

#### 2.1 Study participants and design

The study protocol was approved by the Howard University Human Participants Institutional Review Board, and each subject provided informed consent. A study population of 52 healthy normotensive 18-26 year-old African-American male university

students was included in the study. Criteria for inclusion in the study were: non-smoking status, absence of alcohol abuse (less than two standard alcohol drinks a day), absence of use of medication that could interfere with autonomic modulation, resting systolic/diastolic blood pressure <140/90 mm Hg and body mass index less than 28 kg m<sup>-1</sup>.

### **2.1.1 Paced breathing**

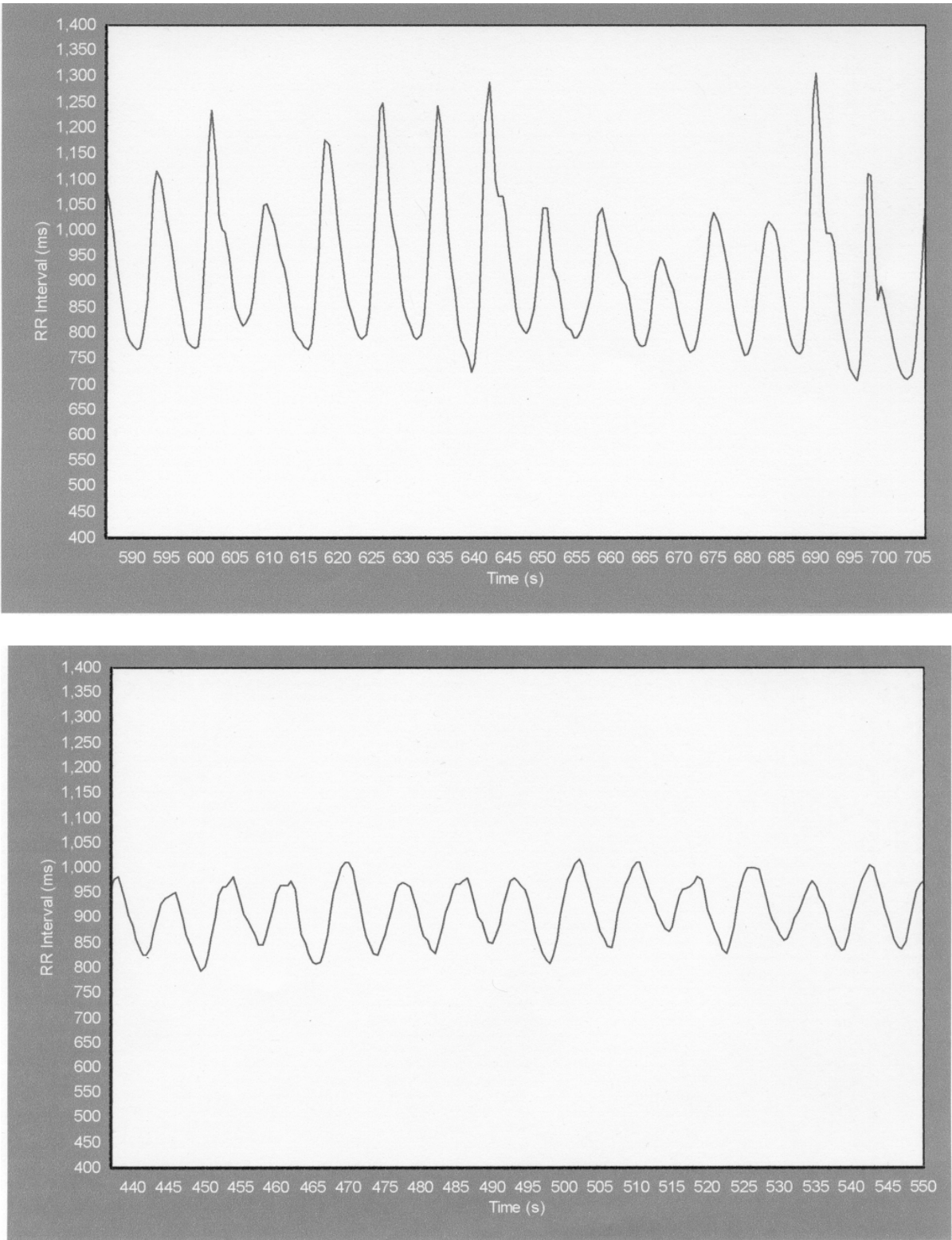
After entering the laboratory subjects were instrumented and instructed as to the experimental procedures. Subjects breathed normally while seated and at rest and 5 min of this resting data was recorded. Following the normal breathing protocol subjects were instructed to perform 5 min of paced breathing in such a manner that each respiratory cycle was 5 s in duration or 12 breaths per min (0.2 Hz). The subject observed a visual tracking image on a computer monitor for periodic durations of inspirations and expirations. Each subject practiced paced breathing for a period of 1 min and was then instructed to perform the paced respiration for 5 min during which time the electrocardiogram signal was recorded using a Biopac MP100 data acquisition system (Biopac Systems, Santa Barbara, CA). The electrocardiogram electrodes were placed on the subject's chest in a standard three-lead position with recordings obtained from lead II.

### **2.1.2 Group assignment**

Two groups of subjects were a priori classified as either "broadband" or "narrowband". The criterion for the "narrowband" group was that the maximum SDNN value, a time-domain marker of vagal modulation, was more than one standard deviation below the mean SDNN for the study population during the 5 min trial of paced breathing. The "broadband" group, thereby, consisted of the subjects exhibiting SDNN one standard deviation less than that of the mean SDNN of the study population. Figure 1 shows cardiogram tracings of representative broadband and narrowband subjects.

### **2.1.3 Heart rate variability analyses**

Heart rate was measured in beats · min<sup>-1</sup> and vagal modulation of HRV in the time domain was measured as the standard deviation of all normal-to-normal standard electrocardiogram inter-beat intervals (SDNN). Time domain HRV, measured as standard deviation of the RR intervals, was expressed in ms and was computed using data acquisition and analysis software specifically designed to measure HRV in the time and frequency domain (Nevrokard, Version 6.3.1, Ljubljana, Slovenia). All time domain HRV data were reported herein as mean SDNN ± standard error. Fast Fourier transform analysis of the electrocardiogram RR intervals was used to spectrally decompose HRV in the frequency domain. For the frequency domain analysis, vagal modulation was represented by the area under the high-frequency power spectrum (HF: 0.14-0.4 Hz) expressed as the power in ms<sup>2</sup>. We also included the LF/HF ratio of heart rate variability as a measure of sympathetic modulation according to the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996). However, this concept has received great criticism (Eckberg, 1999). All time and frequency domain analyses were carried out in accordance with the guidelines put forth by the Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996).



**Fig. 1. Electrocardiogram RR intervals for broadband and narrowband subjects.** Representative cardiogram tracings showing electrocardiogram RR intervals (ms) on the ordinate and time (s) on the abscissa for a representative subject exhibiting characteristics for assignment to the broadband group (Top Panel) and to the narrowband group (Bottom Panel).

2.1.4 Aerobic capacity (VO<sub>2peak</sub>)

The functional work capacity measure of VO<sub>2peak</sub> was performed during a graded exercise treadmill test using the Bruce protocol with stage 1 beginning at an incline of 10% at a speed of 1.7 mph for 3 min. After stage 1, the treadmill incline was increased by 2% and the speed by 0.8 mph every 3 min until voluntary fatigue. Respiratory measures of ventilation and gas fractions of oxygen and carbon dioxide were performed using a Physio-Dyne Max I metabolic system (Physio-Dyne Inc., Quogue, NY). VO<sub>2peak</sub> was defined as the VO<sub>2</sub> value achieved during the last min of the graded treadmill exercise test. Before the graded exercise test of VO<sub>2peak</sub>, the metabolic system was calibrated with known gas concentrations of oxygen and carbon dioxide. The participants performed the VO<sub>2peak</sub> test during the first laboratory visit at the beginning of the study.

2.1.5 Paced and uncontrolled spontaneous breathing conditions

The subjects were trained to breathe at 0.2 Hz by following an analog signal generated by a computer for timing the inspiratory and expiratory phases of respiration. Measurements of heart rate variability made during paced and uncontrolled spontaneous breathing were performed while sitting in a chair.

2.1.6 Mental and nociceptive stress conditions

Mental stress was produced by Stroop word-color conflict testing and physical stress by cold pressor testing using single foot immersion in an ice bath.

2.2 Statistical analysis

The significance of differences in SDNN, the absolute HF power, the LF/HF power was determined by comparing the paced breathing control to the spontaneous breathing condition using Student’s t-test for paired samples. Linear regression analysis was used to verify the correlation between SDNN and HF power. The significance of intergroup (“broadband” vs. “narrowband”) differences was determined using Student’s t-test for independent samples. Probability for all analyses was set at P<0.05. Significance of differences in SDNN across testing conditions was evaluated by analysis of variance (ANOVA), with significance at P<0.05.

3. Results

Table 1 presents the physiological characteristics of the study population showing that it consisted of a healthy group of 18-26 year-old healthy male African-American university students measured during the paced breathing control condition.

Age (years)	20.92 ± 2.48
Weight (kg)	76.03 ± 10.89
Height (cm)	179.44 ± 7.06
Resting Systolic Blood Pressure (mm Hg)	130.08 ± 8.64
Resting Diastolic Blood Pressure (mm Hg)	76.36 ± 9.33
Resting Heart Rate (beats · min <sup>-1</sup> )	75.48 ± 9.65
Peak Oxygen Consumption (mL · kg <sup>-1</sup> · min <sup>-1</sup> )	35.30 ± 7.28

\* Mean ± standard deviation

Table 1. Physiological characteristics of the study population\*



The heart rates of the study population measured during the normal spontaneous breathing condition were found to be lower than those measured during the paced breathing control condition ( $67.9 \pm 1.8$  vs.  $70.1 \pm 1.7$ ,  $P=0.01$ ); this difference was extremely small (3%) and, therefore, not of physiological significance. On the other hand, the heart rates measured during the Stroop word color conflict and cold pressor testing were 16% higher ( $81.3 \pm 2.3$  and  $81.3 \pm 2.3$  vs.  $70.1 \pm 1.7$ ,  $P<0.01$ , respectively) and the heart rates during exercise at 30%-50% of peak oxygen consumption were 34%-77% higher ( $94.5 \pm 2.1$  and  $124.6 \pm 3.5$  vs.  $70.1 \pm 1.7$ ,  $P<0.01$ , respectively) than those measured during the paced breathing control condition; these differences were considered to be physiologically significant.

The study group of 52 subjects was characterized by a maximum SDNN of 249.70 ms, a minimum SDNN of 39.15 ms and a mean  $\pm$  standard deviation of  $108.40 \pm 40.95$  ms during the paced breathing control condition. A subgroup of subjects met the criterion for low HRV (6/52, 12%) and, by experimental design, exhibited a very small SDNN, more than one standard deviation below the mean of the study population, during paced breathing at a fixed frequency (0.2 Hz). This subgroup was differentiated from the larger study group by a minimum SDNN of 39.15 ms, a maximum of 64.55 and a mean  $\pm$  standard deviation of  $49.40 \pm 7.15$  ms ( $P<0.0001$ ).

Figure 1 demonstrates the range of SDNN values measured in this study and shows a significantly greater SDNN of the study population for the paced breathing control condition than for the two conditions of exercise at 30% and 50% of peak oxygen consumption ( $P<0.0001$ ). SDNN measured during the periods of normal spontaneous breathing, at 0.18-0.48 Hz while at rest sitting in a chair and on a cycle ergometer, and Stroop word color conflict testing (mental stress) were smaller than the SDNN measured during the paced breathing control period and intermediate between the largest SDNN during paced breathing and the smallest SDNN measured during aerobic exercise ( $P<0.001$ ). The SDNN measured during cold pressor testing (cold stress) was not significantly different than that during the paced breathing control ( $P>0.1$ ).

Figure 2 shows that the SDNN of the study group during the control paced breathing was significantly greater than that during the uncontrolled spontaneous breathing, both measured while sitting in a chair ( $P<0.05$ ). Figure 3 demonstrates that the group of 6 subjects ("narrowband" group) found to have significantly smaller SDNN of the remaining group of 46 subjects ("broadband" group) during normal spontaneous breathing (0.18-0.4 Hz), both measured at rest while sitting in a chair ( $P<0.05$ ). Figure 4 shows that, between the normal spontaneous breathing and paced breathing conditions, the SDNN of this "low HRV, narrow heart rate bandwidth" or "narrowband" group of 6 subjects increased by  $1.58 \pm 13.59\%$ ; whereas, that of the remaining group of 46 "normal HRV, broad heart rate bandwidth" subjects ("broadband" group) increased by  $30.41 \pm 2.29\%$  ( $P=0.0006$ ) during paced breathing at 0.2 Hz.

Figure 5 presents a comparison of the SDNN of the broad and narrow heart rate bandwidth groups across all of the physiological states studied. By experimental design, the SDNN measured during the paced breathing control condition differentiated between the narrow and broad heart rate bandwidth groups which were also found to be differentiated by the SDNN measured during the normal spontaneous breathing state. These groups were not differentiated by the SDNN measured during the experimental conditions of Stroop word color conflict testing (mental stress), cold pressor testing (cold stress) and exercise at 30% and 50% of peak oxygen consumption.

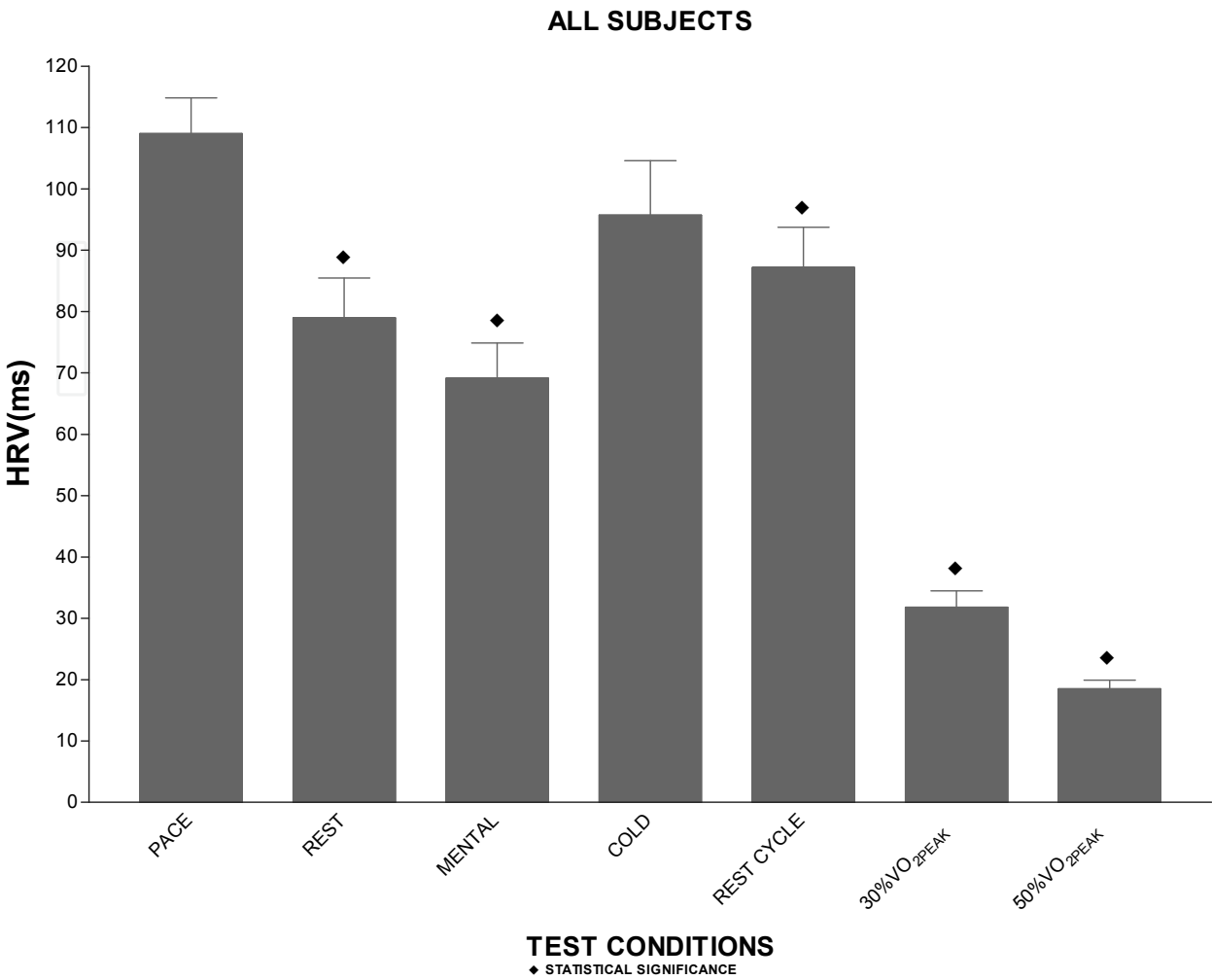


Fig. 2. **Heart rate variability across various testing conditions.** Bars represent heart rate variability (HRV, ms) expressed as time domain measure of mean standard deviation of normal-normal electrocardiogram RR intervals (SDNN) during paced breathing at 0.2 Hz (pace), normal uncontrolled spontaneous breathing at rest sitting in a chair (rest), Stroop word-color conflict testing of mental stress (mental), cold pressor testing of nociceptive stress (cold), normal uncontrolled spontaneous breathing at rest sitting on a cycle ergometer (rest cycle) and aerobic exercise stress at 30% and 50% of peak oxygen consumption (30% VO<sub>2peak</sub>, 50% VO<sub>2peak</sub>). Subjects are 52 healthy young adult African-American males. Data in mean ± standard error. ♦ SDNN different from paced breathing control (pace) at P<0.05.

The “broadband” group’s total spectral power was significantly greater than the “narrowband” group’s total HRV spectral power for paced but not for spontaneous breathing (16,600 ± 1,842 vs. 2,858 ± 176 ms<sup>2</sup>, P=0.01 and 7,807 ± 1,224 vs. 2,106 ± 424 ms<sup>2</sup>, P=0.1, respectively). The “broadband” versus “narrowband” intergroup difference in absolute LF power was not significant for spontaneous breathing. The “broadband” group’s absolute LF power was significantly higher than the “narrowband” group’s absolute LF power (12,830 ± 1,461 vs. 1,981 ± 296 ms<sup>2</sup>, P=0.01) for paced breathing. The “broadband” group’s absolute HF power was not significantly different than the “narrowband” group’s absolute HF power for both paced and spontaneous breathing (P=0.12). The “broadband” versus “narrowband” intergroup difference in the percentages of total LF and HF power was also not significant.

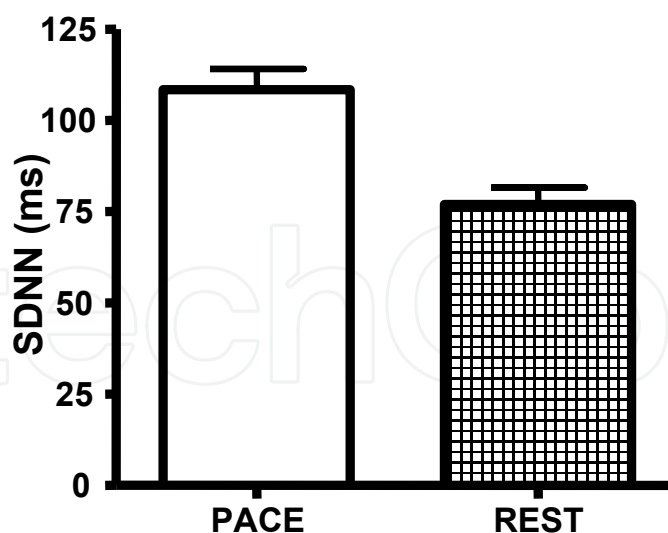


Fig. 3. **Heart rate variability for paced and uncontrolled breathing conditions.** Bars represent heart rate variability (HRV, ms) expressed as time domain measure of mean standard deviation of normal-normal electrocardiogram RR intervals (SDNN) during paced breathing at 0.2 Hz (pace) and normal uncontrolled spontaneous breathing at rest sitting in a chair (rest). Subjects are 52 healthy young adult African-American males. Data in mean  $\pm$  standard error. The rest condition is different from paced breathing control (pace) at  $P < 0.05$ .

Figure 6 shows that the LF/HF ratio of HRV spectral power, a measure of cardiac sympathovagal balance, was significantly higher during paced breathing at 0.2 Hz than during uncontrolled spontaneous breathing for the study group of 52 subjects ( $P < 0.01$ ) and, during paced breathing at 0.2 Hz, was significantly higher for the narrowband band group of 6 subjects than for the broadband group of 46 subjects ( $P < 0.05$ ).

#### 4. Discussion

Humans vary in their responsiveness to environmental stressors and HRV measurements may serve as physiological markers for such variation (DeBecker et al., 1998). HRV in the time domain, measured as standard deviation or standard error, estimates the range of differences in the time intervals between heartbeats (Lucini et al., 2002c). HRV has been used as a measure of autonomic balance that emanates from endogenous sympathetic and parasympathetic rhythms which are partly modulated by respiratory sinus arrhythmia (Hayano et al., 1990; Hrushesky, 1991). Fast Fourier transform (FFT) analysis of HRV differentiates the frequency components of the heart's inter-beat intervals and yields more detailed information about autonomic tone than time domain analysis (Petretta et al., 1995). Such analysis makes it possible to differentiate conditions with physiological features in common. For example, common autonomic contributions to HRV have been evaluated by examining specific FFT frequency bands. This frequency domain analysis is useful under diverse physiological states such as dynamic exercise (Pichon et al., 2004), hypertension associated with sleep apnea (Vanninen et al., 1996; Narkiewicz et al., 1998; Salo et al., 2000) and optic neuropathy (Gutierrez et al., 2002). In this study, we found that healthy young adult African-Americans exhibit greater SDNN, a time domain measure of HRV, during conditions of paced breathing and cold stress than during conditions of normal breathing



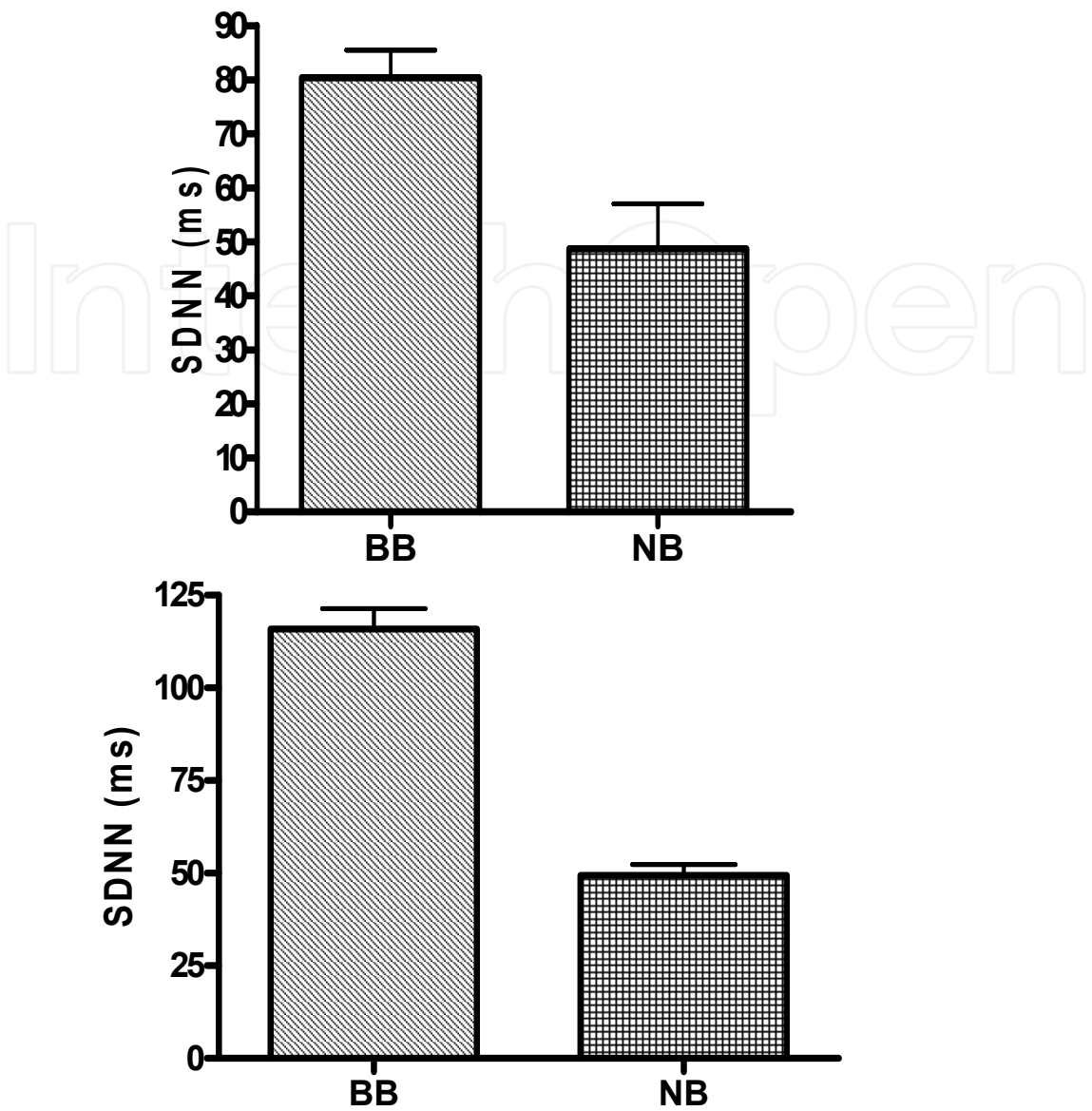


Fig. 4. **Heart rate variability for broadband and narrowband groups.** Bars represent heart rate variability (HRV, ms) expressed as time domain measure of mean standard deviation of normal-normal electrocardiogram RR intervals (SDNN) during paced breathing at 0.2 Hz (Bottom Panel) and normal uncontrolled spontaneous breathing at rest sitting in a chair (Top Panel). Subjects are 46 broadband (BB) healthy young adult African-American males with normal HRV compared to a similar group of 6 narrowband (NB) subjects with low HRV defined as exhibiting SDNN during paced breathing more than one standard deviation from the mean SDNN of the study group of 52 subjects. Data in mean  $\pm$  standard error. The rest condition is different from paced breathing control (pace) at  $P<0.05$ .

and mental stress. We also identified a small subpopulation (12%) of subjects exhibiting SDNN more than one SD below the mean SDNN of the larger study population of 52 males during the paced breathing condition. It was beyond the scope of this part of the study to measure HRV spectral power. Because of the high variability of frequency domain measurements during short time intervals (Taverner et al., 1996), we limited a part of this study to the time domain measurement of SDNN.

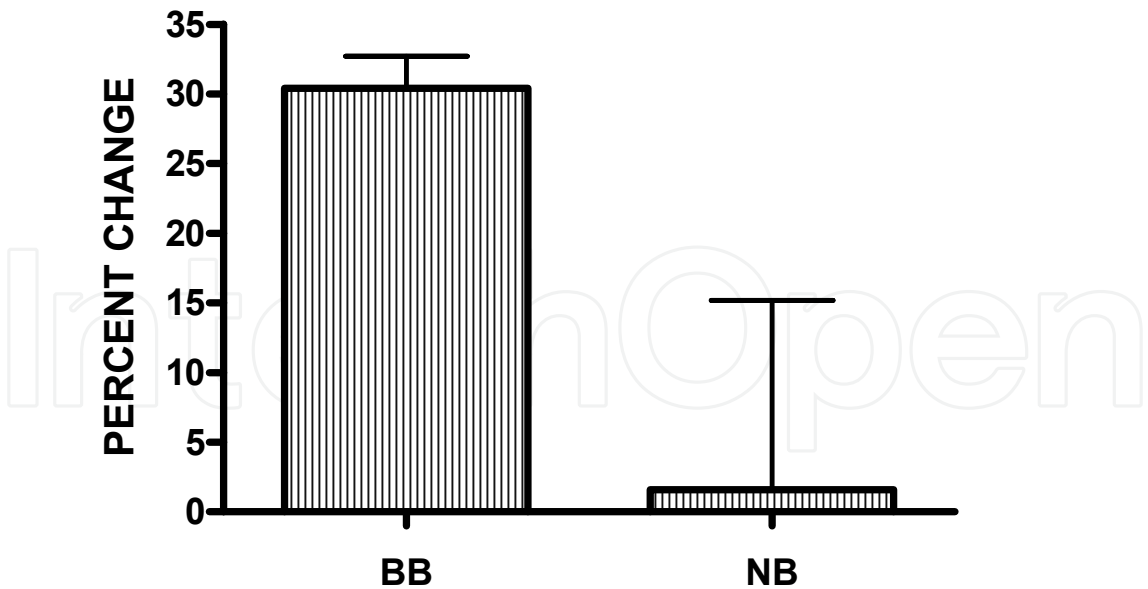


Fig. 5. **Percent change in heart rate variability for broadband and narrowband groups.** Bars represent percent increase in heart rate variability expressed as time domain measure of mean standard deviation of normal-normal electrocardiogram RR intervals (SDNN) during paced breathing at 0.2 Hz. Subjects are 46 broadband (BB) healthy young adult African-American males with normal HRV compared to a similar group of 6 narrowband (NB) subjects with low HRV defined as exhibiting SDNN during paced breathing less than one standard deviation from the mean SDNN of the study group of 52 subjects. Data in mean  $\pm$  standard error. The percent increase in SDNN of the BB group is different from that of the NB group at  $P<0.05$ .

Respiratory sinus arrhythmia is thought to be the main source of HRV. However, there may be other non-autonomic contributions to respiratory sinus arrhythmia and to the high frequency (HF) components of HRV which may distort the signal-to-noise ratio and estimates of capacity for vagal modulation of heart rate (Pichon et al., 2004). HF HRV may mostly reflect noise if breathing shifts a substantial amount of HRV power to the low frequency (LF) range. The low time domain HRV occurring in subjects with apnea syndromes (Vanninen et al., 1996; Narkiewicz et al., 1998; Salo et al., 2000) could also be an effect of respiratory rate and/or tidal volume (Pinna et al., 2006). Low time domain HRV has been found in subjects exhibiting pre-hypertensive (Lucini et al., 2002a) and obesity (Salo et al., 2000) risk factors. Acetylcholine, when released from the vagus nerve, appears to act synergistically with vasoactive intestinal peptide to increase respiratory sinus arrhythmia (Markos and Snow, 2001). Higher HRV spectral frequencies and greater inter-beat intervals have been associated with a high capacity for vagal modulation of heart rate which occurs in normotensive healthy adults breathing at rest (Gutierrez et al., 2002). The peak HRV spectral frequency occurs in the range of HF in normotensive healthy adults breathing at rest and shifts to the range of LF during periods of exercise and stress, as well as, during disease states such as hypertension (Murakami et al., 1996)

Because of the respiration-related variability of electrocardiogram inter-beat (RR) intervals, the necessity of controlling respiratory frequency during measurements of HRV has been demonstrated (De Meersman et al., 1995). We used the frequency of 0.2 Hz during the paced breathing trial because this frequency produced the most reproducible conditions across subjects. As expected, the time domain HRV during paced breathing was significantly greater than during spontaneous breathing in the same subjects. This breathing pattern was

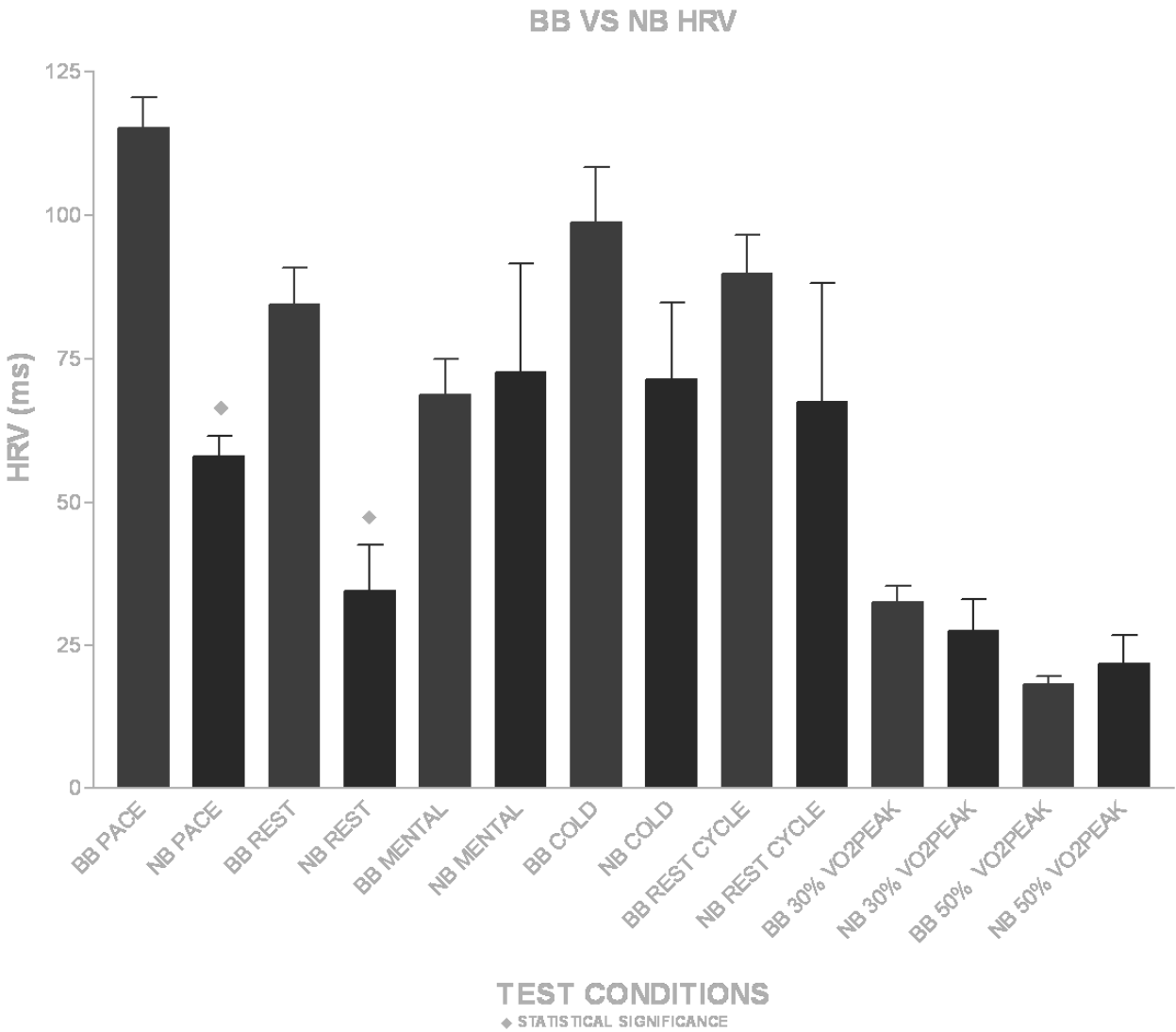


Fig. 6. Heart rate variability across various testing conditions for broadband and narrowband subjects. Bars represent heart rate variability (HRV, ms) expressed as time domain measure of mean standard deviation of normal-normal electrocardiogram RR intervals (SDNN) during paced breathing at 0.2 Hz (pace), normal uncontrolled spontaneous breathing at rest sitting in a chair (rest), Stroop word-color conflict testing of mental stress (mental), cold pressor testing of nociceptive stress (cold), normal uncontrolled spontaneous breathing at rest sitting on a cycle ergometer (rest cycle) and aerobic exercise stress at 30% and 50% of peak oxygen consumption (30%  $VO_{2peak}$ , 50%  $VO_{2peak}$ ). Subjects are 46 broadband (BB) healthy young adult African-American males with normal HRV compared to a similar group of 6 narrowband (NB) subjects with low HRV defined as exhibiting SDNN during paced breathing more than one standard deviation from the mean SDNN of the study group of 52 subjects. Data in mean  $\pm$  standard error. ♦ NB different from BB at  $P<0.05$ .

under technical supervision and in the low range of normal respiratory rate. Breathing in the range of 3-9 breaths per minute (0.05-0.15 Hz) may produce higher amplitude respiratory sinus arrhythmia because of more complete acetylcholine metabolism during exhalation (Song and Lehrer, 2003). Respiratory sinus arrhythmia might also be maximized if subjects breathe at frequencies controlled by other physiological processes. For example, when breathing between 4-7 breaths per minute (0.07-0.12 Hz), the baroreceptor reflex might be stimulated; thereby, causing a resonance effect and an increase in HRV (Vaschillo et al., 2002). In the present study, paced breathing was associated with significantly lower SDNN (0.2 Hz) than the spontaneous breathing trials (0.18-0.48 Hz) and we did not measure tidal volume which has been shown to positively modulate the high frequency (vagal) component of HRV spectral power (Pinna et al., 2006). At a high respiratory frequency paced breathing has been shown to produce predominance of the HF power of HRV in normal healthy adult subjects during meditation (Cysarz and Bussing, 2005). HRV studied during paced breathing in subgroups of pre-hypertensive and hypertensive middle-aged men and women has been shown to produce greater HF power in the pre-hypertensive than in the hypertensive subgroups (Prakash et al., 2005).

HRV in a subgroup of healthy normotensive middle-aged persons whose natural spontaneous respiratory frequency ranged 9-27 breaths per minute (0.15-0.45 Hz) with predominance of the HF power of HRV was compared to another subgroup whose natural respiratory frequency was less than 9 breaths per min (0.15 Hz) (Pinna et al., 2006). In that study, paced breathing at 15 breaths per min (0.25 Hz) failed to alter either the time domain or the frequency domain HRV parameters. In a study population of healthy adults, data sets matching respiratory frequencies at rest with those during dynamic exercise demonstrated that the LF and HF powers of HRV were not changed by controlled breathing in the absence of dynamic exercise but were significantly decreased at the same respiratory frequencies during exercise (Bartels et al., 2004). Because of the expected transients and uncertainties about the role of respiration and significance of changes in frequency domain HRV, we limited this part of the study to use of a time domain measure of HRV that, based on the current knowledge, would be more easily interpreted.

## 5. Conclusions

This study has demonstrated a wide range of SDNN measurements performed under various experimental conditions from the largest SDNN measured during paced breathing at 0.2 Hz to the smallest during exercise at 30%-50% of peak oxygen consumption in a group of 52 healthy young adult African-American males. The SDNN measured during short periods of normal spontaneous breathing at 0.18-0.48 Hz while at rest sitting both in a chair and on a cycle ergometer, cold stress and mental stress were intermediate, between those of paced breathing and aerobic exercise. SDNN was used to differentiate a "narrowband" subgroup (6/52 subjects, 12%) on the basis of a much smaller observed difference in SDNN between paced and normal spontaneous breathing and, despite an increase in the heart rate, the absence of a decrement in SDNN during a state of mental stress. These findings suggest that mental stress, elicited by Stroop word conflict testing, seems to have a differentiating effect on SDNN, an easily performed and interpreted time domain measure of heart rate variability. In the frequency domain, higher LF/HF heart rate variability spectral power, a reliable measure of sympathetic modulation of the heart rate, differentiated the paced from the uncontrolled spontaneous breathing condition and the same "narrowband" subgroup, as demonstrated in figures 7 and 8.

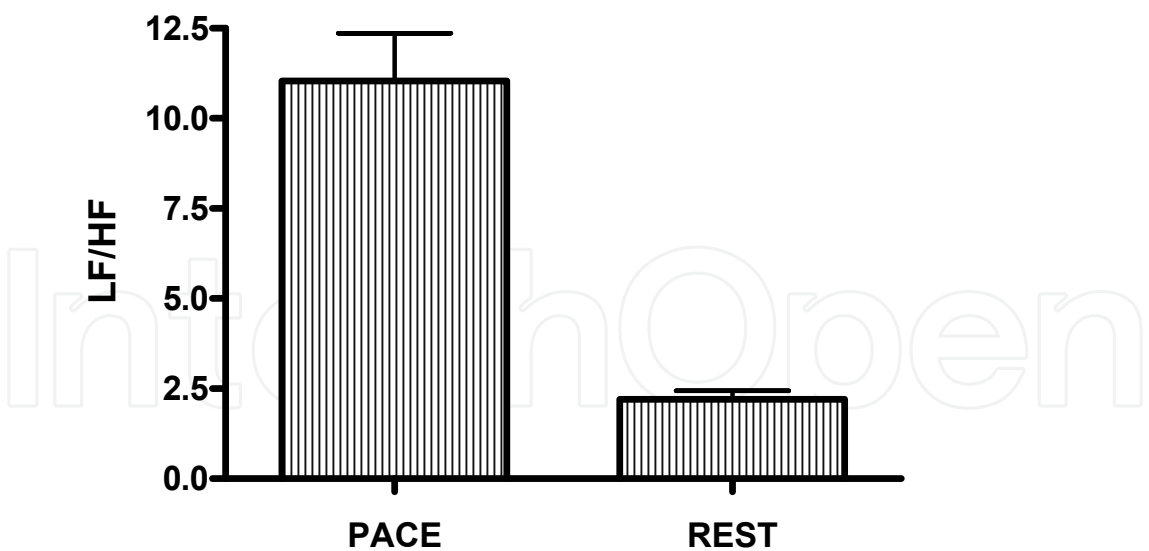


Fig. 7. Heart rate variability spectral power measure of sympathovagal balance for paced and uncontrolled breathing conditions. Bars represent low frequency/high frequency ratio (LF/HF) of heart rate variability spectral power measured by fast Fourier transform analysis of electrocardiogram RR intervals during paced breathing at 0.2 Hz (pace) and normal uncontrolled spontaneous breathing at rest sitting in a chair (rest). Subjects are 52 healthy young adult African-American males. Data in mean  $\pm$  standard error. The rest condition is different from paced breathing control (pace) at  $P<0.01$ .

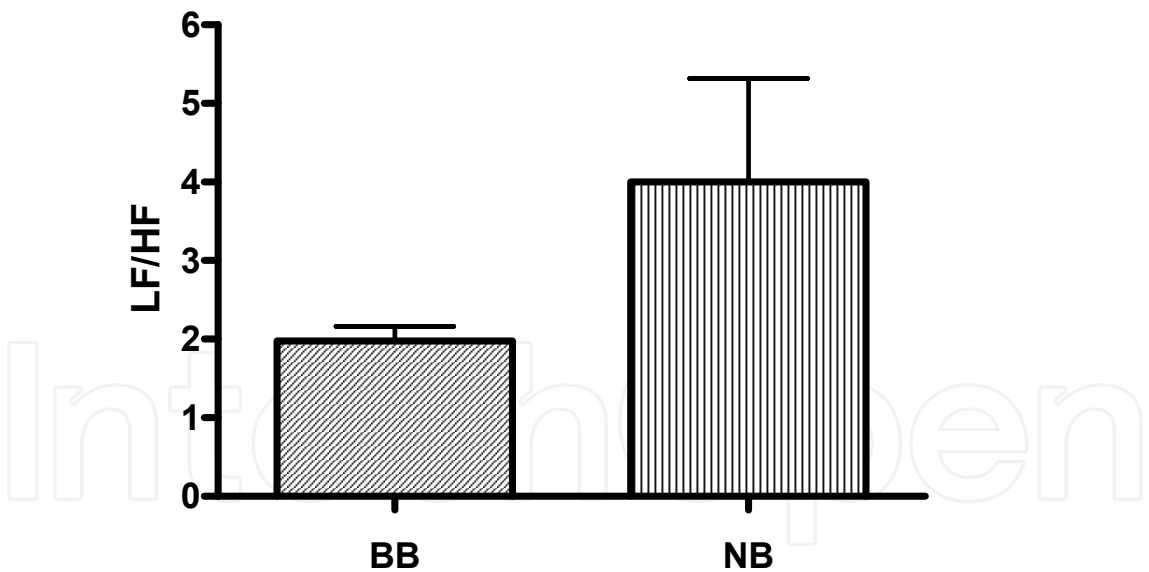


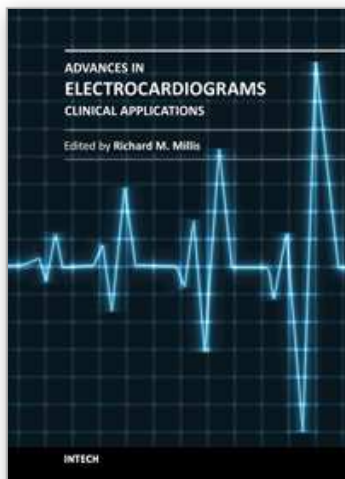
Fig. 8. Heart rate variability spectral power measure of sympathovagal balance for broadband and narrowband groups. Bars represent low frequency/high frequency ratio (LF/HF) of heart rate variability spectral power measured by fast Fourier transform analysis of electrocardiogram RR intervals during paced breathing at 0.2 Hz. Subjects are 46 broadband (BB) healthy young adult African-American males with normal HRV compared to a similar group of 6 narrowband (NB) subjects with low HRV defined as exhibiting SDNN during paced breathing more than one standard deviation from the mean SDNN of the study group of 52 subjects. Data in mean  $\pm$  standard error. LF/HF of the BB group is different from that of the NB group at  $P<0.05$ .



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## **Advances in Electrocardiograms - Clinical Applications**

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Electrocardiograms have become one of the most important, and widely used medical tools for diagnosing diseases such as cardiac arrhythmias, conduction disorders, electrolyte imbalances, hypertension, coronary artery disease and myocardial infarction. This book reviews recent advancements in electrocardiography. The four sections of this volume, Cardiac Arrhythmias, Myocardial Infarction, Autonomic Dysregulation and Cardiotoxicology, provide comprehensive reviews of advancements in the clinical applications of electrocardiograms. This book is replete with diagrams, recordings, flow diagrams and algorithms which demonstrate the possible future direction for applying electrocardiography to evaluating the development and progression of cardiac diseases. The chapters in this book describe a number of unique features of electrocardiograms in adult and pediatric patient populations with predilections for cardiac arrhythmias and other electrical abnormalities associated with hypertension, coronary artery disease, myocardial infarction, sleep apnea syndromes, pericarditides, cardiomyopathies and cardiotoxicities, as well as innovative interpretations of electrocardiograms during exercise testing and electrical pacing.

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